05:00

725-5 Improved Outcome in Patients with Prior Percutaneous Transluminal Coronary Angioplasty and an Evolving Myocardial Infarction

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Despite the increasing prevalence of PTCA, the outcome of patients with prior PTCA who present with an MI and are treated with thrombolytic therapy is unknown. We compared the outcome of the 1647 patients in GUSTO who had undergone a prior PTCA with the 39,150 patients who had not undergone a prior PTCA. Patients with a prior PTCA had a higher incidence of previous MI (67 vs 14%) and CABG (19 vs 4%), lower ejection fraction (50 vs 52%), less anterior MI (34 vs 39%), and lower systolic bp (127.6 vs 129.1). In addition, their mean age was lower (60.4 vs 61.2 yrs), and time to treatment was shorter (2.9 vs 3.1 hrs). Outcomes were:

	Prior PTCA	No Prior PTCA	P-value
24 hr mortality (%)	1.8	2.7	0.03
30 day mortality (%)	5.6	7.0	0.04
CHF (%)	14.3	16.3	0.02
Re-infarction (%)	4.6	4.0	NS
Ischemia (%)	24.0	19.8	<0.0001
CABG (%)	12.2	8.5	<0.00001
PTCA (%)	56.2	32.1	<0.00001

Conclusion: Patients with prior PTCA had a lower 24-hr and 30-day mortality and less CHF compared with patients without prior PTCA. Although they experienced more recurrent ischemia, no increase in re-infarction was observed. The protective effect of prior PTCA may be due to reactive changes in the treated coronary segment and is associated with a more frequent revascularization.

05:15

725-6 Persistent Intramyocardial Segmental Dysfunction After Anterior Infarction Treated with Reperfusion and ACE Inhibition

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We have previously demonstrated intramyocardial segmental dysfunction throughout the LV in the first week after anterior myocardial infarction. We used magnetic resonance tagging (MRI) to determine the evolution in regional intramyocardial function and LV mass index (LVMI), end-diastolic and end-systolic volumes (EDV, ESV), and ejection fraction (EF) during the first 8 weeks after anterior MI in patients treated with reperfusion and ACE inhibitors (ACEI). We studied 10 normals (7 male, 3 female, ages 43 ± 14) and 8 patients (7 male, age 52 ± 16) after first anterior MI with peak CK 2871 ± 2244 with MRI at day 6 ± 2 (week 1) and day 58 ± 10 (week 8) post-MI. All patients had isolated LAD disease and were reperfused with tPA (n = 1), PTCA (n = 3), or both (n = 4). All were treated with ACEI and 5 with β-blockers at both time points. Breath-hold, segmented k-space, TurboFlash, tagged images with the subject prone on an elliptical spine coil were obtained throughout systole with 7 mm thick short axis slices (n = 11 ± 2) spanning the LV from apex to base. Global parameters did not change over the 8 week period (mean ± S.D.):

	Week 1	Week 8	p
LVMI (g/m^2)	114 ± 22	118 ± 9	NS
EDV (ml)	87 ± 27	108 ± 24	NS
ESV (ml)	55 ± 18	64 ± 29	NS
EF (%)	36 ± 11	42 ± 16	NS

Percent intramyocardial circumferential end-systolic segment shortening (%S) was measured at 3 transmural locations in each of the 4 regions on each LV short axis slice. Mean %S ± SD was compared by long axis location:

	Normals	Pts. (Week 1)	Pts. (Week 8)
Apex (%)	23.0 ± 2.9	6.3 ± 6.3*	10.0 ± 3*#
Mid-ventricle (%)	20.5 ± 2.6	8.6 ± 4.4*	11.3 ± 4.3*
Base (%)	17.0 ± 4.9	11.3 ± 3.7*	13.5 ± 3.2†

*$p < 0.02$ vs. normals, #$p < 0.02$ vs. 1 week, †$p = 0.07$ vs. normals

Significant improvement is limited to the apex, which includes infarcted and adjacent noninfarcted myocardium. In conclusion, while reperfusion and ACE inhibition blunt remodeling after anterior infarction, intramyocardial segmental function remains depressed throughout the left ventricle.

726 Primary Heart Muscle Disease — Myocarditis, Biopsy

Monday, March 20, 1995, 4:00 p.m.–5:30 p.m.
Ernest N. Morial Convention Center, Room 6

04:00

726-1 Expression of Inducible Nitric Oxide Synthase in the Myocardium of Acute Myocarditis — A Serial Cardiac Biopsy Study

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In endotoxin shock, hypotension and impaired myocardial contractility may be caused by increased nitric oxide (NO) formed by inducible NO synthase (iNOS) which is induced by cytokines and bacterial lipopolysaccarides.

We have investigated whether iNOS is expressed in serially biopsied samples from the right ventricle of 20 cases of acute myocarditis by immunohistochemistry and in situ hybridization. Distinct iNOS immunoreactivity was observed in cardiomyocytes, endothelial cells, vascular smooth muscle cells and macrophages in all cases during acute active stage, in which cardiac function was severely impaired. Immunoelectron microscopic distribution of iNOS was evident in the cytosol of these cells. However, little staining was disclosed in the myocardium during the convalescent stage, showing normal cardiac function. The distribution of myocytes with elevated iNOS mRNA concentration was identical to that of these immunoreactive cells. *Conclusions.* The present study shows the expression of human myocardial iNOS in acute myocarditis, suggesting that the enhanced production of NO by iNOS is cytotoxic and accounts, in part, for myocardial injury and reduced myocardial contractility during acute illness.

04:15

726-2 The Role of Myocardial Catecholamine: Promising or Proved in Diagnosis and Prognosis of Primary Heart Muscle Diseases?

Petar M. Seferović, Stepan Stepanović, Ružica Maksimović, Miodrag Ostojić, Slavko Simeunović, Jovan D. Vasiljević, Dragana Seferović, Arsen Ristić, Dragan V. Simić, Vesna Dangubić. *Institute for Cardiovascular Diseases of the University Medical Center, Belgrade, Yugoslavia*

The role of myocardial catecholamine concentration (MCC) in diagnosis and outcome of primary heart muscle diseases (HMD) is still incompletely defined. To elucidate this problem we analyzed diagnostic utility of MCC measurements in pts with biopsy proven myocarditis (BPM), hypertrophic cardiomyopathy (HCM), and idiopathic dilated cardiomyopathy (DCM), as well as MCC prognostic impact in DCM. Our study group consisted of 86 pts, 20 of them with BPM (80% males, aged 18–42 yrs), 34 HCM pts (64% males, aged 29–54 yrs) and 32 pts with DCM (75% males, aged 21–56 yrs). At the initial assessment all pts underwent cardiac catheterization and endomyocardial biopsy (EMB). Myocardial norepinephrine (MNEC), epinephrine (MEC), and dopamine (MDC) concentrations were measured in EMB samples using catechol-O-methyl-transferase radioenzymatic method. Obtained values (ng/g of fresh myocardial tissue ft) are shown in the table below:

	MNEC	MEC	MDC
BPM	415.4 ± 71.1†	57.3 ± 4.8†	76.6 ± 9.2 ns
HCM	781.0 ± 125.1**	91.3 ± 13.1**	78.1 ± 9.3 ns
DCM	262.2 ± 68.9‡	36.9 ± 7.1‡	72.6 ± 12.1 ns

†-$p < 0.01$ (BPM vs. DCM); **-$p < 0.01$ (HCM vs. BPM); ‡-$p < 0.01$ (HCM vs. DCM)

In addition, we analyzed the effect of MNEC and MEC on the five years survival of 31 DCM pts. Survival (18/31) was significantly better in pts with higher MNEC (411.8 ± 43.4 ng/g ft survivors vs. 278.5 ± 75.7 ng/g ft non-survivors, $p < 0.01$) and MEC (55.3 ± 4.5 ng/g ft survivors vs. 39.2 ± 9.3 ng/g ft non-survivors, $p < 0.01$). Univariate and multivariate analysis demonstrated that both MNEC and MEC predicted long-term survival independently and their low concentrations were associated with increased mortality.

In conclusion, these data indicate that MCC measurements may be helpful as a complementary tool in diagnostic evaluation of pts with HMD. Furthermore, MNEC and MEC should be considered as valuable prognostic markers of the long-term survival of DCM pts.

MONDAY PM